REMARKS

Applicant's counsel thanks the Examiner for the careful consideration given the application. In view of the Examiner's objections raised against the open formulation of pending claim 1, applicant has determined to amend the claims limited to the specific sense and anti-sense PNA molecules disclosed in the Application as filed. These PNA molecules are SEQ ID NO: 1 and SEQ ID NO: 3.

SEQ ID NO: 1 (5'-TCCACCCAGCGCGTCC-3') corresponds to the anti-sense PNA molecule which is complementary to a unique sequence of 5'-UTR region of N-myc gene.

SEQ ID NO: 3 (5'-ATGCCGGGCATGATCT-3') corresponds to the sense anti-gene PNA sequence which is complementary to a sequence of N-myc gene exon-2.

Amended claim 1 finds support in the specification and in the original claims (claims 2 and 6), wherein SEQ ID NO: 1 and SEQ ID NO: 3 have been disclosed (see pages 7-9 of the description), their capacity to inhibit N-myc gene expression in medical models and their therapeutic efficacy in N-myc expressing neuroblastoma treatments have been properly demonstrated.

Claim Rejections – 35 U.S.C. Section 103

The claims have been rejected under Section 103(a) as being unpatentable over Sun, et al. and Cutrona, et al.

Sun, et al refers to a method to improve PNA cellular up-take involving anti-sense PNA conjugation to somatostatin analogs. Sun, et al demonstrates that the up-take of anti-sense PNA molecules, administered in cells expressing somatostatin receptors, is improved when PNAs are conjugated to somatostatin analogs. In fact, they use this approach in cell lines overexpressing N-myc gene (such as IMR32) and they find that, when anti-sense PNAs, conjugated to somatostatin analogs and directed against N-myc gene, are administered to cells, N-myc expression is compromised.

Cutrona, et al demonstrates how the conjugation of PNAs to Nuclear Localization Signal (NLS) peptides (above all PKKKRKV peptide) helps cell nuclei penetration. In particular, Cutrona, et al synthesizes an NLS-linked anti-gene PNA complementary to a unique sequence of the second exon of c-myc. They administer the specific PNA to Burkitt's lymphoma cell lines

(these cells are characterized by c-myc hyper-expression resulting from chromosomal translocation) and they find that NLS-linked PNAs are able to block the expression of the targeted gene (for example c-myc). Therefore, they are able to inhibit the biological functions of the desired gene.

The scope of the cited references is different with respect to the purpose of the pending Application, nevertheless, applicant has taken into account the Examiner's observations and has decided to limit claim 1 to SEQ ID NO:1 and SEQ ID NO:3.

Neither Cutrona, et al, nor Sun, et al, taken alone or in combination, discloses or suggests SEQ ID NO:1 and SEQ ID NO:3 to inhibit N-myc gene expression and, therefore, it is clear that amended claim 1, now limited to SEQ ID NO:1 and SEQ ID NO:3, is novel and non-obvious.

Moreover, applicant submits that amended claim 1 is commensurate in scope with the specification, wherein the capacity in N-myc expression inhibition of SEQ ID NO:1 and SEQ ID NO:3 has been well demonstrated.

In view of all the foregoing, it is clear that the claims as now limited are not anticipated or obvious in view of the cited prior art. For these reasons, applicant submits that the claims are now in condition for allowance, which is respectfully requested.

If any further fees are required by this communication, please charge such fees to our BUG5-38919.

Respectfully submitted,
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